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Cardiotoxicity in haematological diseases: are the tyrosine kinase inhibitors imatinib and nilotinib safe?

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Introduction: Chemotherapy-induced cardiotoxicity is a growing concern. The true cardiotoxic impact of new drugs such as tyrosine kinase inhibitors is unknown, especially the ones used for chronic myeloid leukaemia. We aim to evaluate nilotinib and imatinib induced cardiotoxicity.
Methods: Single-center prospective study of consecutive patients with chronic myeloid leukaemia treated with tyrosine kinase inhibitors during 2015. Patients underwent an initial clinical, laboratorial and echocardiographic evaluation, repeated one year after therapy initiation.
Results: Eleven patients were included [60.0 (11) years, 63.6% of males; 7 patients treated with imatinib and 4 with nilotinib]. After one year of follow-up, all patients remained in functional NYHA class I, with a similar Minnesota quality of life score [21 (20) vs. 21 (19), p = NS]. Also there was no difference in the biomarkers evaluated: cystatin-C [0.9 (0.2) vs. 0.8 (0.2) mg/L, p = NS; NT-proBNP 46.0 (45.0) vs. 42.0 (34.0) pg/mL, p = NS]. Previous to the TKI treatment, all patients had normal left ventricular ejection fraction (LVEF) [(median 67% (63–69)], without structural abnormalities. During the follow-up, there weren't differences regarding the LVEF, left atrium volume, E/A ratio, deceleration time, septal e', lateral e', E/e' ratio and tricuspid annular plane systolic excursion. With regard to myocardial deformation, all patients presented normal values of longitudinal, circumferential and radial strain in the baseline study, without changes during follow-up [DML -21.3 (6, 1) vs. -21.7 (6.0)%, p = NS; DMC -20.0 (9.3) vs. -22.3 (5.3)%, p = NS; DMR 36.9 (21.3) vs. 39.2 (19.2)%, p = NS]. In addition, there were no differences between the two tyrosine kinase inhibitors used, considering all the aforementioned variables.
Conclusion: No clinical, laboratory or echocardiographic evidence of nilotinib and imatinib induced cardiotoxicity was observed, even when myocardial deformation analysis was performed. However, these results should be confirmed in larger studies, ideally multicentre, given the low incidence of chronic myeloid leukaemia.